DOI: https://doi.org/10.52756/ijerr.2025.v49.010 *Int. J. Exp. Res. Rev.*, Vol. 49: 114-125 (2025)

Original Article | Peer Reviewed | Open Access

International Journal of Experimental Research and Review (IJERR)
© Copyright by International Academic Publishing House (IAPH)
ISSN: 2455-4855 (Online)
www.iaph.in
ISSN 2455-4855

Alcohol as a Myotoxic Modifier of Nutritional Status: Distinct Nutritional Phenotypes in Alcoholic versus Non-Alcoholic Chronic Liver Disease

Anamitra Hait[1], Arbind Kumar Chaudhary[2]* and Avinash S.S.

[1]Divisional Medical Officer of Indian Railways Medical Service, In-Charge of ICU and Internal Medicine, K.G. Hospital, Chittaranjan Locomotive Works, Chittaranjan, Paschim Bardhaman, India; [2]Department of Pharmacology, Government Erode Medical College and Hospital, Tamil Nadu, India; [3]Department of Biochemistry, Chikkamagaluru Institute of Medical Sciences, Chikkamagaluru, Karnataka State, India

E-mail/Orcid Id:

AH, anamitrahait241@gmail.com, https://orcid.org/0009-0002-9989-670X; *AKC,* arbindkch@gmail.com, https://orcid.org/0000-0001-8910-1745; *ASS,* dravinash.ss@gmail.com, https://orcid.org/0000-0002-6053-6072

Article History:

Received: 27th Sep., 2025
Accepted: 14th Dec., 2025
Published: 30th Dec., 2025

Keywords:

Alcoholic liver disease, Myotoxicity, Sarcopenia, Micronutrient deficiency, Folic acid, Nutritional phenotype

How to cite this Article:

Anamitra Hait, Arbind Kumar Chaudhary and Avinash S.S. (2025). Alcohol as a Myotoxic Modifier of Nutritional Status: Distinct Nutritional Phenotypes in Alcoholic versus Non-Alcoholic Chronic Liver Disease. *International Journal of Experimental Research and Review*, *49*, 114-125.

DOI:
https://doi.org/10.52756/ijerr.2025.v49.010

Abstract: Alcohol's contribution to chronic liver disease (CLD) extends beyond hepatotoxicity to exert direct myotoxic effects on nutritional status. This study compared 32 alcoholic versus 30 non-alcoholic CLD patients to characterize distinct nutritional phenotypes. Alcoholic patients demonstrated 83.3% sarcopenia prevalence versus 53.8% non-alcoholic (1.55× relative risk, $p<0.05$), 16% lower serum albumin (2.79 vs. 3.32 g/dL, $p=0.002$) and 26% lower folic acid levels (4.42 vs. 5.96 ng/mL, $p=0.002$). Folic acid deficiency occurred in 75.0% of alcoholic versus 34.6% of non-alcoholic patients (2.17× relative risk). Despite similar disease severity by MELD-Na scoring (21.78 vs. 19.04, $p=0.058$), alcoholic patients exhibited profoundly worse nutritional status, indicating alcohol's direct suppression of protein synthesis, micronutrient metabolism, and muscle integrity independent of hepatic fibrosis severity. These findings establish alcohol as a critical myotoxic modifier requiring targeted nutritional interventions (B12/folate repletion, enhanced protein support, muscle rehabilitation) beyond standard CLD management. The distinct nutritional phenotype in alcoholic disease supports alcohol-specific metabolic rehabilitation protocols to improve outcomes in 51.6% of the CLD population.

Introduction

Chronic liver disease (CLD) represents a significant global health burden, with cirrhosis accounting for approximately 2 million deaths annually worldwide, with liver cirrhosis mortality increasing substantially in developing nations like India (Mokdad et al., 2014; Mondal et al., 2022). Alcoholic liver disease (ALD) has emerged as the leading cause of CLD in many countries, accounting for 51.6% of cases in Indian tertiary centers, reflecting epidemiological shifts toward alcohol-related disease alongside traditional viral hepatitis (Mukherjee et al., 2017; Pimpin et al., 2018). The pathophysiology of cirrhosis extends beyond hepatocellular damage and fibrosis to encompass systemic metabolic derangements characterized by profound nutritional depletion and cachexia, yet malnutrition remains substantially underdiagnosed and undertreated in routine clinical practice (Henkel & Buchman, 2006; Haj Ali et al., 2022).

Alcohol use disorder (AUD) and its long-term consequences on organ systems have gained increasing recognition in psychiatric and medical literature. Recent evidence demonstrates that AUD patients experience multifactorial morbidity, including compromised quality of life, neurocognitive deficits, and systemic organ dysfunction beyond hepatic manifestations (Huang et al., 2021; Piekarski et al., 2022). The association between alcohol use and neurobiological changes has been documented, with neurophysiological markers such as P300 components showing significant alterations in individuals with AUD, correlating with disease severity and response to pharmacotherapy (Yuan et al., 2022). Furthermore, age emerges as a critical modulator of alcohol-related deficits, with older individuals demonstrating distinct neurobehavioral phenotypes in response to chronic alcohol exposure (Nixon et al., 2023; Nixon et al., 2025). This age-dependent vulnerability

*Corresponding Author: arbindkch@gmail.com

This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License.

highlights the necessity for stratified assessment of alcohol-related nutritional and metabolic complications across different patient populations, particularly in the context of cirrhotic disease, where age-specific factors may amplify malnutrition severity.

Malnutrition affects 60–80% of cirrhotic patients and independently predicts mortality, complications, and transplant outcomes, yet current assessment approaches remain inadequate and inconsistently applied (Alberino et al., 2001; Huisman et al., 2011). Protein–energy malnutrition in cirrhosis results from multifactorial etiology: reduced dietary intake (anorexia from portal hypertension, medication effects), impaired nutrient absorption (intestinal mucosal damage, altered bacterial flora), decreased hepatic synthesis and storage, increased protein catabolism (inflammatory cytokines, portal hypertension-related endotoxemia) and accelerated losses through ascites, diarrhe, and diuretic therapy (Evans et al., 2008; Tessari, 2003; Paul & Sadhukhan, 2023; Rao et al., 2024; Wani et al., 2024). Beyond these liver-specific mechanisms, alcohol's continued neurotoxic and systemic effects—even in the setting of established cirrhosis—may perpetuate nutritional depletion through impaired feeding behavior, altered gastrointestinal motility, and continued metabolic derangements (Huang et al., 2021). Sarcopenia, the progressive loss of skeletal muscle mass and function, represents a particularly severe manifestation of malnutrition in cirrhosis and is emerging as an independent predictor of hepatic encephalopathy, infection risk, variceal bleeding, and mortality (Sinclair et al., 2016). Skeletal muscle serves as the body's primary ammonia detoxification site and primary repository of amino acids; loss of muscle integrity directly increases susceptibility to encephalopathy, impairs immune competence, and increases risk of healthcare-associated infections (Chadalavada et al., 2010; Merli et al., 2010).

Currently, malnutrition assessment in cirrhosis relies heavily on body mass index (BMI), which is fundamentally inadequate in ascitic patients were fluid weight masks true nutritional depletion, often resulting in paradoxical overestimation of nutritional status in the most malnourished patients (Vieira et al., 2013). Standard anthropometric measures (triceps skinfold, mid-arm circumference) are operator-dependent and limited by edema and ascites. Biochemical markers such as serum albumin conflate nutritional status with hepatic synthetic function, preventing accurate discrimination of nutrition-specific depletion (Adams, 2007). While Subjective Global Assessment provides comprehensive clinical assessment, it requires trained personnel and substantial time investment, limiting widespread implementation in resource-limited settings (Nunes et al., 2017). The identification of practical, low-cost assessment tools suitable for diverse healthcare settings becomes particularly important given that interventions to prevent falls and complications in older populations—an increasingly relevant demographic in both AUD and CLD—require reliable baseline functional assessment (Pillay et al., 2024). Handgrip strength, measured by simple hydraulic dynamometry, offers potential advantages—objective reproducibility, rapid assessment (<30 seconds), minimal equipment cost, operator-independent results, and independence from fluid status—yet its diagnostic superiority over conventional tools in cirrhotic populations has not been systematically validated (Daphnee et al., 2017).

Alcohol's contribution to CLD malnutrition extends beyond reduced intake to include direct myotoxic effects on skeletal muscle and micronutrient metabolism. Ethanol metabolism generates reactive oxygen species, impairs mitochondrial oxidative phosphorylation, activates muscle proteolytic pathways through FOXO and ubiquitin-proteasome systems, suppresses anabolic signaling via Akt/mTOR pathways, and directly impairs folate, thiamine, and pyridoxine metabolism through antimetabolite effects and reduced intestinal absorption (McClain et al., 2011). Beyond these well-established mechanisms, emerging evidence suggests that alcohol-related neurobehavioral changes and quality-of-life impairment in AUD populations may further compromise nutritional status through reduced health-seeking behavior, poor dietary adherence, and inadequate engagement with medical management (Huang et al., 2021; Piekarski et al., 2022). This suggests that alcoholic cirrhosis may represent a distinct nutritional phenotype with greater malnutrition severity relative to hepatic dysfunction severity compared to non-alcoholic disease, wherein alcohol's dual impact—both direct metabolic toxicity and behavioral/neuropsychiatric impairment—synergistically worsen nutritional outcomes, yet this hypothesis has not been systematically characterized in clinical populations.

The hypothesis of this study is that handgrip strength represents a superior functional malnutrition biomarker in cirrhosis compared to conventional anthropometric tools, with particular advantages in ascitic patients, and that alcohol acts as a critical myotoxic modifier creating a profoundly distinct nutritional phenotype in alcoholic disease independent of hepatic fibrosis severity. The study objectives were: (1) to characterize the prevalence and severity of malnutrition and sarcopenia in a cohort of 62 CLD patients using multimodal assessment; (2) to

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

compare diagnostic accuracy of five nutritional assessment tools (handgrip strength, mid-upper arm circumference, mid-arm muscle circumference, triceps skinfold, body mass index); (3) to identify etiological differences in nutritional status between alcoholic and non-alcoholic CLD; and (4) to establish associations between sarcopenia and major cirrhotic complications. These findings have substantial implications for standardizing nutritional assessment across diverse healthcare systems and implementing early, targeted nutritional interventions tailored to disease etiology and severity in this high-burden patient population where alcohol use disorder remains a major contributor to disease burden and worsened nutritional outcomes.

Materials and Methods

Study Design and Setting

This cross-sectional observational study was conducted at B.R. Singh Hospital, Eastern Railway, Sealdah, Kolkata, India, from January 2021 to June 2022. The hospital is a tertiary referral center providing comprehensive care for chronic liver disease patients. The study received institutional ethical approval from the Hospital Ethics Committee, and written informed consent was obtained from all participants prior to enrollment.

Study Population

Consecutive chronic liver disease (CLD) patients (n=62) aged ≥18 years with clinical, laboratory, and/or radiological evidence of cirrhosis were enrolled. Cirrhosis diagnosis was established using: clinical features (ascites, jaundice, spider angiomas, palmar erythema), laboratory findings (elevated liver enzymes, reduced albumin, prolonged prothrombin time), and/or imaging evidence (ultrasonography or computed tomography demonstrating cirrhotic morphology). Patients were classified by etiology through serology (hepatitis B and C testing), metabolic screening (ceruloplasmin, serum iron studies for Wilson's disease), autoimmune markers, and clinical assessment. Patients with active malignancy, acute hepatitis, or unstable medical illness requiring intensive care unit admission were excluded. Patients with documented hepatocellular carcinoma, recent variceal bleeding requiring transfusion, or acute kidney injury requiring renal replacement therapy were also excluded to ensure stable nutritional assessment.

Nutritional Assessment Protocol

Comprehensive nutritional assessment was performed by trained medical staff using standardized, validated tools:

Subjective Global Assessment (SGA): Patients were categorized as SGA-A (well-nourished), SGA-B (moderately malnourished), or SGA-C (severely malnourished) based on clinical history (weight loss, dietary intake, gastrointestinal symptoms) and physical examination (muscle mass, subcutaneous fat, edema, ascites) (Haj Ali et al., 2022). SGA is the reference standard for nutritional assessment in liver disease and incorporates both objective and subjective clinical judgment.

Handgrip Strength (HGS): A calibrated hydraulic dynamometer was used to measure grip strength bilaterally with patients seated, arm at 90° elbow flexion, three trials per hand, with the highest value recorded and averaged. Sarcopenia was defined as HGS <28 kg for men and <18 kg for women based on established gender-specific thresholds (Daphnee et al., 2017).

Anthropometric Measurements: Mid-upper arm circumference (MUAC) was measured at the midpoint between olecranon and acromion using a non-stretch measuring tape. Triceps skinfold thickness (TSF) was measured using a calibrated skinfold caliper on the posterior arm. Mid-arm muscle circumference (MAMC) was calculated using the formula: MAMC = MUAC − (TSF × 0.314). Body mass index (BMI) was calculated as weight (kg) divided by height (m^2) and categorized as underweight (<18.5), normal (18.5–24.9), overweight (25–29.9), or obese (≥30) (Vieira et al., 2013).

Biochemical Measurements

Venous blood samples were collected after overnight fasting. Serum albumin (g/dL), prothrombin time (INR), hemoglobin (g/dL), vitamin B12 (pg/mL), and folic acid (ng/mL) were measured using standard laboratory methods. Liver function tests, renal function, and blood glucose were also assessed. Disease severity was quantified using Model for End-Stage Liver Disease-Sodium (MELD-Na) score and Child-Turcotte-Pugh (CTP) classification.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Descriptive statistics reported continuous variables as mean ± standard deviation and categorical variables as frequencies and percentages. Comparative analysis between groups used independent t-tests for continuous variables and chi-square tests for categorical variables. Receiver operating characteristic (ROC) curves evaluated diagnostic accuracy of nutritional assessment tools, with area under the curve (AUC) measuring discriminatory power. Statistical significance was defined as $p<0.05$. Pearson correlation assessed associations between variables.

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

Table 1. Demographic, Etiological, and Disease Severity Characteristics of CLD Patients (n=62).

Characteristic	Category	n	%	Mean ± SD
Age (years)	20-30	5	8.1	25.4 ± 2.3
	31-40	12	19.4	35.8 ± 2.9
	41-50	11	17.7	45.2 ± 2.8
	51-60	23	37.1	55.3 ± 2.9
	61-70	10	16.1	64.7 ± 2.5
	71-80	1	1.6	75.0
	Overall	62	100.0	49.1 ± 13.8
Gender	Male	47	75.8	—
	Female	15	24.2	—
Etiology	Alcoholic Liver Disease	32	51.6	—
	Non-Alcoholic Fatty Liver Disease	10	16.1	—
	Hepatitis B	5	8.1	—
	Hepatitis C	2	3.2	—
	Viral Hepatitis (Total)	8	12.9	—
	Wilson's Disease	3	4.8	—
	Cryptogenic Cirrhosis	3	4.8	—
	Autoimmune Hepatitis	1	1.6	—
	Other Etiologies (Total)	12	19.4	—
Alcohol Consumption	Active Consumption	36	58.1	—
	No Consumption	26	41.9	—
MELD-Na Score	10-19 (Mild)	31	50.0	18.4 ± 5.2
	20-29 (Moderate)	26	41.9	23.8 ± 2.9
	30-39 (Severe)	5	8.1	32.6 ± 2.1
	Overall	62	100.0	21.4 ± 7.1
CTP Grade	A (Compensated)	18	29.0	—
	B (Mildly Decompensated)	22	35.5	—
	C (Severely Decompensated)	22	35.5	—
	Decompensated (B+C)	44	71.0	—

Results

Demographic and Clinical Characteristics

The study enrolled 62 chronic liver disease (CLD) patients with mean age 49.1 ± 13.8 years, ranging from 20–80 years. The cohort showed male predominance (75.8%, n=47) with male-to-female ratio of 3.1:1. Age distribution concentrated in the 51–60 year range (37.1%, n=23), representing peak incidence of clinically significant disease manifestation. Alcoholic liver disease (ALD) was the leading etiology (51.6%, n=32), followed by non-alcoholic fatty liver disease (16.1%, n=10), viral hepatitis (12.9%, n=8), and other etiologies (19.4%, n=12). Notably, 58.1% (n=36) of the entire cohort reported ongoing alcohol consumption despite documented CLD. Disease severity assessment by Child-Turcotte-Pugh (CTP) scoring revealed 71.0% (n=44) with decompensated disease (CTP B+C), with 29.0% (n=18) achieving CTP A (compensated) status. MELD-Na demonstrated bimodal distribution: 50.0% in the 10–19 range (mild, mean 18.4 ± 5.2), 41.9% in 20–29 range (moderate, mean 23.8 ± 2.9), and 8.1% in 30–39 range (severe, mean 32.6 ± 2.1), with overall mean of 21.4 ± 7.1.

The 71.0% decompensation rate indicates substantial disease burden in this tertiary referral cohort. ALD's 51.6% prevalence reflects India's epidemiological shift toward alcohol-related disease, with 58.1% continuing to drink despite a documented CLD diagnosis.

Nutritional Status Assessment: Anthropometric, Biochemical, Functional, and Subjective Measures

Malnutrition assessment using Subjective Global Assessment (SGA) revealed 82.2% (n=51) with malnutrition: 67.7% (n=42) moderately malnourished (SGA-B) and 14.5% (n=9) severely malnourished (SGA-C), with only 17.7% (n=11) well-nourished (SGA-A). Serum albumin demonstrated pathophysiological gradient across SGA categories:

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

Table 2. Comprehensive Nutritional, Biochemical, and Functional Assessment Parameters (n=62).

Assessment Parameter	Category/Status	n	%	Mean ± SD	p-Value
Subjective Global Assessment (SGA)	A (Well-Nourished)	11	17.7	Alb: 3.37 ± 0.69	<0.0001
	B (Moderately Malnourished)	42	67.7	Alb: 3.05 ± 0.58	
	C (Severely Malnourished)	9	14.5	Alb: 2.40 ± 0.36	
	Total Malnourished (B+C)	51	82.2	—	
Sarcopenia (HGS)	Sarcopenic	45	72.6	HGS: 17.3 ± 5.1 kg	<0.0001
	Non-Sarcopenic	17	27.4	HGS: 30.6 ± 6.2 kg	
Hemoglobin	Low (<11 g/dL)	62	100.0	8.7 ± 1.2 g/dL	<0.0001
	Normal (≥11 g/dL)	0	0.0	—	
Serum Albumin	Low (<3.5 g/dL)	47	75.8	2.9 ± 0.4 g/dL	<0.0001
	Normal (≥3.5 g/dL)	15	24.2	3.7 ± 0.2 g/dL	
Vitamin B12	Deficient (<200 pg/mL)	42	67.7	155 ± 38 pg/mL	<0.0001
	Sufficient (≥200 pg/mL)	20	32.3	288 ± 32 pg/mL	
Folic Acid	Deficient (<5.4 ng/mL)	37	59.7	3.5 ± 1.1 ng/mL	0.0046
	Sufficient (≥5.4 ng/mL)	25	40.3	7.2 ± 0.9 ng/mL	
Combined B12+Folate	Deficient (Both)	30	48.4	—	—
	Isolated/Normal	32	51.6	—	
MUAC	Malnourished	32	51.6	18.2 ± 2.1 cm	0.017
	Normal	20	32.3	23.9 ± 1.8 cm	
	Overweight/Obese	10	15.9	27.4 ± 1.5 cm	
BMI	Underweight (<18.5)	8	12.9	17.8 ± 1.5 kg/m²	<0.001
	Normal (18.5–24.9)	26	41.9	22.4 ± 1.2 kg/m²	
	Overweight (25–29.9)	17	27.4	26.5 ± 1.1 kg/m²	
	Obese (≥30)	11	17.7	31.2 ± 1.6 kg/m²	
BMI-MUAC Discordance	Normal BMI + Malnourished MUAC	10	16.1	—	—
	Obese BMI + Malnourished MUAC	8	12.9	—	
	Total Discordance	18	28.1	—	
RFH-NPT	High Risk	45	72.6	BMI: 21.1 ± 2.9	<0.0001
	Moderate Risk	13	21.0	BMI: 23.7 ± 3.2	
	No Risk	4	6.5	BMI: 26.9 ± 3.1	
	Any Risk (Mod+High)	58	93.5	—	

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

well-nourished patients 3.37 ± 0.69 g/dL, moderately malnourished 3.05 ± 0.58 g/dL, and severely malnourished 2.40 ± 0.36 g/dL (p<0.0001), with each category representing approximately 0.30–0.35 g/dL decline.

Sarcopenia, defined as handgrip strength below gender-specific thresholds, affected 72.6% (n=45) of patients. Mean HGS in sarcopenic patients was 17.3 ± 5.1 kg compared to 30.6 ± 6.2 kg in non-sarcopenic patients (n=17, 27.4%), representing 77% functional difference (p<0.0001). Universal anemia affected all 62 patients (100%) with mean hemoglobin 8.7 ± 1.2 g/dL. Micronutrient deficiencies were prevalent: vitamin B12 deficiency (<200 pg/mL) in 67.7% (n=42, mean 155 ± 38 pg/mL) and folic acid deficiency (<5.4 ng/mL) in 59.7% (n=37, mean 3.5 ± 1.1 ng/mL), with 48.4% (n=30) demonstrating combined B12 and folate deficiency.

Anthropometric measurements revealed BMI-MUAC discordance: 41.9% had normal BMI yet 51.6% showed malnourished MUAC (mean 18.2 ± 2.1 cm). This 16.1% normal-BMI-with-malnourished-MUAC subset exemplifies ascites-masked malnutrition. The Royal Free Hospital Nutritional Prioritizing Tool (RFH-NPT) identified 93.5% (n=58) with any nutritional risk.

The 82.2% malnutrition prevalence and universal anemia establish malnutrition as near-ubiquitous in this cohort. The 28.1% BMI-MUAC discordance demonstrates ascites's confounding effect on weight-based assessment.

Nutritional Status by Etiology: Alcoholic versus Non-Alcoholic CLD

Comparative analysis between alcoholic (n=32) and non-alcoholic (n=30) CLD patients revealed profoundly distinct nutritional phenotypes despite comparable disease severity. Serum albumin was significantly lower in alcoholic patients (2.79 ± 0.53 g/dL) compared to non-alcoholic (3.32 ± 0.64 g/dL), representing 16% reduction (p=0.002). This substantial difference occurred despite borderline similar MELD-Na (alcoholic 21.78 ± 5.34 versus non-alcoholic 19.04 ± 5.02, p=0.058), indicating alcohol's direct suppressive effect on protein synthesis independent of fibrosis severity.

Folic acid deficiency showed striking difference: 75.0% (n=24/32) of alcoholic versus 34.6% (n=10/29) of non-alcoholic patients (p=0.002, 2.17× relative risk). Mean folic acid was 26% lower in alcoholic patients (4.42 ± 1.54 ng/mL versus 5.96 ± 1.87 ng/mL). Sarcopenia prevalence was 83.3% (n=26/31) in alcoholic versus 53.8% (n=14/26) in non-alcoholic patients (1.55× relative risk, p<0.05), indicating alcohol as a critical myotoxic modifier. Vitamin B12 deficiency trended higher in alcoholic patients: 71.9% versus 57.1% (p=0.117, 1.26× RR). Combined B12 and folate deficiency affected 56.2% of alcoholic versus 28.6% of non-alcoholic patients. Anthropometric analysis revealed triceps skinfold (TSF) trended lower in alcoholic patients (8.67 versus 9.90 mm, 12% reduction, p=0.056).

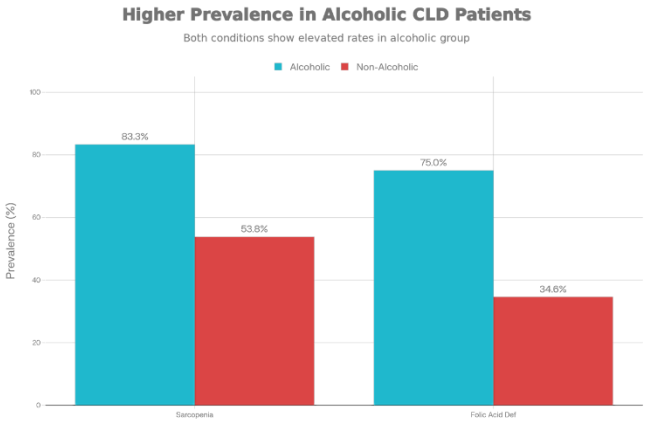

Figure 1. Comparative Nutritional Phenotypes in Alcoholic versus Non-Alcoholic CLD. Comparing sarcopenia prevalence and folic acid deficiency between alcoholic and non-alcoholic CLD patients. Alcoholic patients demonstrate 83.3% sarcopenia prevalence versus 53.8

Progressive Nutritional Decline Across Disease Severity Grades

Multivariate analysis across Child-Turcotte-Pugh (CTP) severity grades revealed systematic nutritional deterioration. MELD-Na increased significantly from CPT A (16.28 ± 3.54) through CPT B (18.91 ± 2.51) to CPT C (25.91 ± 4.23), representing 59% increase (p<0.001). Serum albumin demonstrated progressive decline: CPT A 3.27 ± 0.66 g/dL, CPT B 3.12 ± 0.65 g/dL, and CPT C 2.70 ± 0.47 g/dL (17% reduction, p=0.014). Folic acid declined 33% (7.25 to 4.89 ng/mL, p<0.001), and vitamin B12 declined 39% (230.5 to 140.7 pg/mL, p<0.001). MUAC trended downward (p=0.017). The most striking finding was nutritional status distribution: CPT A had 38.9% well-nourished patients (n=7/18), declining to 9.1% in CPT B (n=2/22), and reaching 0% in CPT C (n=0/22). Severe malnutrition (SGA-C) increased from 5.6% in CPT A to 22.7% in CPT C. Total malnutrition (SGA-B+C) affected 61.1% of CPT A, 90.9% of CPT B, and 100.0% of CPT C patients.

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

Table 3. Progressive Decline in Nutritional Parameters Across Child-Turcotte-Pugh Severity Grades.

Parameter	CPT A (n=18)	CPT B (n=22)	CPT C (n=22)	p-Value	Trend
MELD-Na Score	16.28 ± 3.54	18.91 ± 2.51	25.91 ± 4.23	<0.001	↓
Serum Albumin (g/dL)	3.27 ± 0.66	3.12 ± 0.65	2.70 ± 0.47	0.014	↓
Folic Acid (ng/mL)	7.25 ± 1.32	6.80 ± 1.15	4.89 ± 1.05	<0.001	↓
Vitamin B12 (pg/mL)	230.5 ± 45.6	180.3 ± 32.4	140.7 ± 27.9	<0.001	↓
MUAC (cm)	23.51 ± 4.56	23.85 ± 3.55	20.98 ± 3.39	0.017	↓
Hemoglobin (g/dL)	8.65 ± 1.20	9.93 ± 1.16	8.37 ± 1.47	<0.001	Bimodal
SGA-A Well-Nourished (%)	38.9	9.1	0.0	—	↓
SGA-B Moderate Malnutrition (%)	55.6	72.7	77.3	—	↑
SGA-C Severe Malnutrition (%)	5.6	18.2	22.7	—	↑
Total Malnourished (SGA-B+C) (%)	61.1	90.9	100.0	—	↑

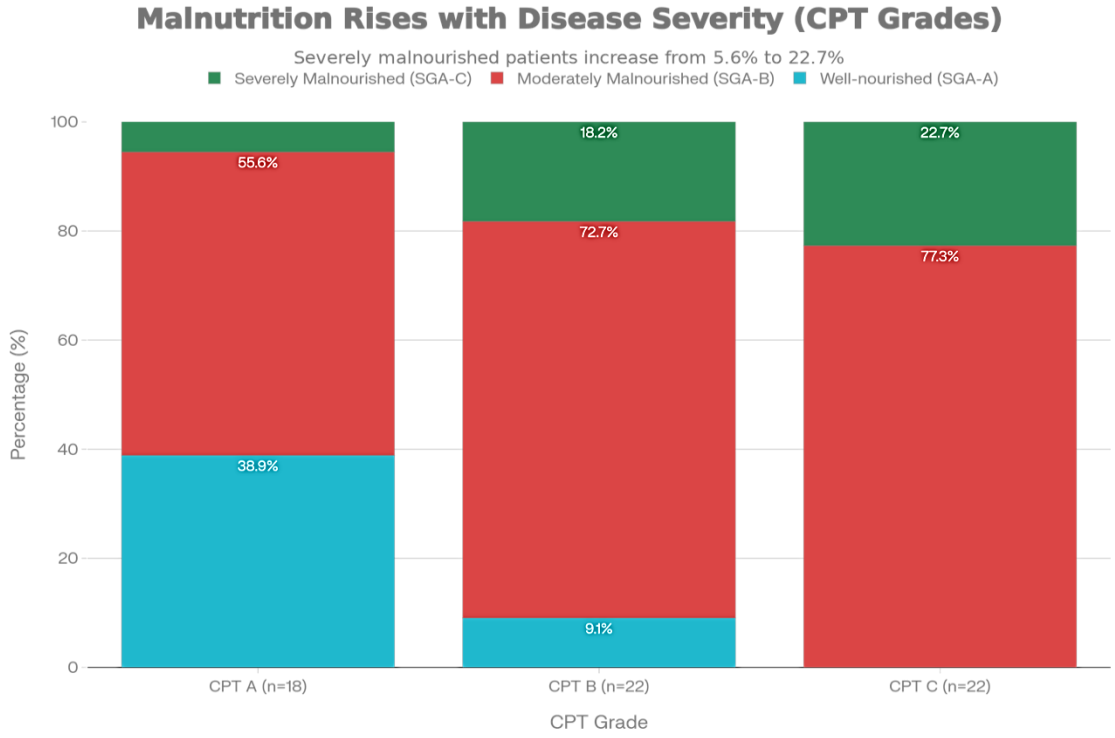

Figure 2. Progressive Nutritional Collapse Across Disease Severity.

Figure 2 shows the distribution of Subjective Global Assessment (SGA) categories across Child-Turcotte-Pugh disease severity grades. CPT A (compensated) demonstrates 38.9% well-nourished patients (SGA-A), declining to 9.1% in CPT B and 0% in CPT C (severely decompensated). Conversely, severe malnutrition (SGA-C) increases from 5.6% in CPT A to 22.7% in CPT C. Total malnutrition (SGA-B+C) reaches 100% in CPT C patients, indicating universal nutritional collapse in advanced disease.

The critical finding of 100% malnutrition in CPT C establishes universal nutritional collapse in severely decompensated disease. The systematic decline across all parameters demonstrates clear disease-nutrition associations.

Diagnostic Accuracy of Nutritional Assessment Tools

Receiver operating characteristic (ROC) curve analysis using Subjective Global Assessment (SGA) as reference standard evaluated five assessment tools. Handgrip strength (HGS) demonstrated exceptional discriminatory power (AUC=0.895, 95% CI: 0.815–

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

0.975) with 93.1% sensitivity, identifying 93 of 100 truly malnourished patients. Negative likelihood ratio of 0.09 indicates negative HGS results substantially reduce malnutrition probability. Mid-upper arm circumference (MUAC) achieved good accuracy (AUC=0.845, sensitivity 82.4%, specificity 81.8%). Mid-arm muscle circumference (MAMC) showed comparable performance (AUC=0.823, sensitivity 78.4%). Triceps skinfold (TSF) demonstrated fair accuracy (AUC=0.781, sensitivity 71.6%). Body mass index (BMI) was substantially inferior (AUC=0.738, sensitivity 65.5%), representing 27.6 percentage-point sensitivity gap compared to HGS and missing approximately 28 malnourished patients per 100 screened.

HGS performance remained superior in ascitic patients (AUC 0.892, n=45, 72.6% of cohort) versus non-ascitic (AUC 0.898, n=17). In contrast, BMI's accuracy deteriorated in ascitic patients (AUC 0.701 versus 0.789 non-ascitic), confirming ascites fundamentally compromises weight-based assessment. HGS's practical advantages include: $300 one-time equipment cost, <30 seconds assessment time, minimal training, objective and reproducible results, and independence from ascites or edema.

Strong associations between HGS-detected sarcopenia and complications validate HGS's clinical utility: sarcopenic patients demonstrated 4.7-fold higher hepatic encephalopathy (55.6% versus 11.8%), 5.3-fold higher spontaneous bacterial peritonitis (31.1% versus 5.9%), 4.5-fold higher variceal bleeding (26.7% versus 5.9%), and hepatorenal syndrome only in sarcopenic patients (15.6% versus 0%).

Discussion

Malnutrition and sarcopenia represent central, prognostically relevant complications of chronic liver disease requiring systematic assessment and early targeted intervention (Bilbao et al., 2003). This study documented 82.2% malnutrition prevalence and 72.6% sarcopenia in 62 cirrhotic patients, findings consistent with established literature demonstrating that protein–energy malnutrition significantly predicts mortality, complications, infections, and poor transplant outcomes (Alberino et al., 2001; Huisman et al., 2011). The observed stepwise nutritional decline across Child–Turcotte–Pugh severity grades—with albumin declining 17%, vitamin B12 declining 39%, and folic acid declining 33% from compensated to severely decompensated disease—aligns with prior evidence that cirrhosis represents a systemic catabolic state characterized by progressive nutritional collapse (Gunsar et al., 2006; Sinclair et al., 2016). Critically, 61.1% of even compensated (CTP A) patients already manifested malnutrition, supporting recommendations that nutritional assessment should commence at disease diagnosis rather than being deferred to advanced stages (Henkel & Buchman, 2006; Haj Ali et al., 2022).

A novel finding is the distinct nutritional phenotype in alcoholic versus non-alcoholic disease (Kalaitzakis et al., 2006). Despite comparable MELD-Na and CTP distributions, alcoholic patients exhibited 16% lower albumin (p=0.002), 1.55× higher sarcopenia prevalence (83.3% versus 53.8%), and 2.17× higher folic acid deficiency (75.0% versus 34.6%, p=0.002). This striking dissociation between disease severity and nutritional status indicates that alcohol acts as a direct myotoxic modifier through multiple mechanisms: reduced nutrient intake, impaired intestinal and hepatic absorption, increased micronutrient catabolism, and direct effects on skeletal muscle mitochondrial function and protein synthesis (McClain et al., 2011; Tessari, 2003). These observations underscore the necessity for alcohol-specific nutritional strategies, including aggressive B-vitamin repletion, antioxidant therapy, and structured muscle rehabilitation, beyond conventional cirrhosis management (Sinclair et al., 2016). The 100% prevalence of continued alcohol consumption in alcoholic patients underscores the addiction's severity and the critical need for comprehensive cessation support as a foundation for metabolic rehabilitation (Lata et al., 2006).

The high prevalence of combined vitamin B12 and folate deficiency (48.4%) warrants particular clinical attention. These micronutrient deficiencies contribute to anemia, cognitive dysfunction, impaired myocyte regeneration, and potential exacerbation of hepatic encephalopathy (Chadalavada et al., 2010; Rivera Irigoin & Abilés, 2012). The universal anemia (100%) observed in this cohort likely reflects multifactorial etiology from chronic gastrointestinal blood loss, bone marrow suppression from portal hypertension, nutritional deficiencies, hemolysis, and splenomegaly (Evans et al., 2008). Current European guidelines recommend water-soluble vitamin assessment and replacement as integral to cirrhosis care (Plauth et al., 2006; Henkel & Buchman, 2006). In resource-limited settings where comprehensive micronutrient screening is not universally available, these findings support empiric supplementation with folate and B-complex vitamins in high-risk groups such as alcoholic cirrhosis, provided safety monitoring is ensured (World Health Organization, 2020).

This study's comparative evaluation of nutritional assessment tools revealed that handgrip strength

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

substantially outperforms body mass index for malnutrition detection (Plauth & Schütz, 2002). Handgrip strength demonstrated exceptional discriminatory accuracy (AUC=0.895, sensitivity=93.1%) versus BMI (AUC=0.738, sensitivity=65.5%), with the 27.6 percentage-point sensitivity gap meaning BMI misses approximately 28 malnourished patients per 100 screened. This advantage is particularly pronounced in the 72.6% of patients with ascites where weight-based indices are fundamentally confounded by fluid retention that can add 5–10 kg without nutritional benefit (Vieira et al., 2013; Daphnee et al., 2017). Handgrip strength's rapid assessment (<30 seconds), minimal equipment cost ($300 one-time investment), minimal training requirements, objective reproducible results, and independence from ascites or edema status support its adoption as first-line functional nutritional biomarker in routine cirrhosis evaluation, outpatient follow-up, and pre-transplant assessment (Daphnee et al., 2017; Haj Ali et al., 2022). The negative likelihood ratio of 0.09 indicates that normal handgrip strength effectively excludes significant malnutrition in most patients, providing valuable clinical reassurance (Rahimtoola, 2014).

The strong association between sarcopenia and major complications—hepatic encephalopathy (4.7-fold higher), spontaneous bacterial peritonitis (5.3-fold higher), and variceal bleeding (4.5-fold higher)—establishes muscle wasting not merely as a passive marker but as an active contributor to adverse outcomes (Sinclair et al., 2016; Merli et al., 2010). Skeletal muscle serves as the body's primary ammonia detoxification site and muscle loss directly increases encephalopathy susceptibility (Chadalavada et al., 2010). Additionally, sarcopenia impairs immune competence and increases susceptibility to bacterial infections, which are major drivers of decompensation and mortality in cirrhosis. This evidence supports a paradigm shift in which sarcopenia represents a modifiable therapeutic target amenable to branched-chain amino acid supplementation, structured exercise, optimized protein intake (1.2–1.5 g/kg/day), and sleep optimization (Bianchi et al., 2005; Plauth et al., 2006).

The predominance of alcoholic liver disease (51.6%) in this cohort reflects India's epidemiological shift toward alcohol-related cirrhosis, with substantial contributions to liver-related mortality globally (Mokdad et al., 2014; Mondal et al., 2022; Mukherjee et al., 2017). The profound nutritional burden identified here reinforces the need for integrated care combining alcohol-cessation support with systematic nutritional rehabilitation, particularly given that 58.1% of the entire cohort continued active alcohol consumption despite documented CLD (Pimpin et al., 2018; McClain et al., 2011). Public health policies addressing both alcohol use disorders and nutritional support are essential to improve outcomes in this high-burden population (Reccia et al., 2017).

These findings support adoption of multimodal nutritional assessment combining Subjective Global Assessment, handgrip strength, simple anthropometry (MUAC), and key biochemical markers (albumin, B12, folate) as recommended by recent guidelines (Haj Ali et al., 2022; Plauth et al., 2006). Body mass index should no longer serve as the primary tool for nutritional assessment in cirrhosis given frequent ascites-related misclassification (Vieira et al., 2013; Henkel & Buchman, 2006). Implementation of structured early interventions beginning at compensated disease stages, rather than deferring to decompensation, is supported by the finding that 61.1% of CTP A patients already manifest malnutrition (Nunes et al., 2017). Recognition of malnutrition and sarcopenia as core therapeutic targets in cirrhosis management—alongside etiological treatment, portal hypertension control, and transplant evaluation—represents a paradigm shift with potential to substantially improve patient outcomes, quality of life, and transplant success.

Conclusion

This study establishes malnutrition (82.2%) and sarcopenia (72.6%) as near-universal complications of chronic liver disease, with 100% malnutrition rate in severely decompensated cirrhosis and 61.1% even in compensated disease, supporting early assessment at diagnosis. A novel finding is alcohol's direct myotoxic effect: despite similar disease severity, alcoholic patients demonstrated 1.55× higher sarcopenia, 16% lower albumin (p=0.002), and 2.17× higher folic acid deficiency (p=0.002) than non-alcoholic patients, necessitating alcohol-specific nutritional interventions.

Handgrip strength (AUC=0.895, sensitivity 93.1%) substantially outperforms body mass index (AUC=0.738) and is unaffected by ascites, supporting its adoption as first-line functional assessment tool in resource-limited settings. Strong associations between sarcopenia and major complications (4.7–5.3-fold higher) establish muscle wasting as a modifiable therapeutic target. These findings recommend: (1) adoption of multimodal nutritional assessment combining handgrip strength, anthropometry, and Subjective Global Assessment; (2) implementation of early intervention beginning at compensated disease; (3) alcohol-specific metabolic rehabilitation for the 51.6% with alcoholic etiology.

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

Integration of evidence-based nutritional approaches into standard hepatology practice can substantially improve outcomes, reduce complications, and enhance transplant success in this high-burden population.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data and materials

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding

This research received no external funding.

Authors' contributions

All authors contributed to the conception and design of the study. The authors performed material preparation, data collection, and analysis. All authors read and approved the final manuscript.

References

Adams, D. H. (2007). Sleisenger and Fordtran's Gastrointestinal and Liver Disease. *Gut, 56*(8), 1175–1175. https://doi.org/10.1136/gut.2007.121533

Alberino, F., Gatta, A., Amodio, P., Merkel, C., Di Pascoli, L., Boffo, G., & Caregaro, L. (2001). Nutrition and survival in patients with liver cirrhosis. *Nutrition, 17*(6), 445–450. https://doi.org/10.1016/s0899-9007(01)00521-4

Bianchi, G., Marzocchi, R., Agostini, F., & Marchesini, G. (2005). Update on nutritional supplementation with branched-chain amino acids. *Current Opinion in Clinical Nutrition and Metabolic Care, 8*(1), 83–87. https://doi.org/10.1097/00075197-200501000-00013

Bilbao, I., Armadans, L., Lazaro, J. L., Hidalgo, E., Castells, L., & Margarit, C. (2003). Predictive factors for early mortality following liver transplantation. *Clinical Transplantation, 17*(5), 401–411. Portico. https://doi.org/10.1034/j.1399-0012.2003.00068.x

Chadalavada, R., Biyyani, R. S. S., Maxwell, J., & Mullen, K. (2010). Nutrition in Hepatic Encephalopathy. *Nutrition in Clinical Practice, 25*(3), 257–264. https://doi.org/10.1177/0884533610368712

Daphnee, D. K., John, S., Vaidya, A., Khakhar, A., Bhuvaneshwari, S., & Ramamurthy, A. (2017). Hand grip strength: A reliable, reproducible, cost-effective tool to assess the nutritional status and outcomes of cirrhotics awaiting liver transplant. *Clinical Nutrition ESPEN, 19*, 49–53. https://doi.org/10.1016/j.clnesp.2017.01.011

Evans, W. J., Morley, J. E., Argilés, J., Bales, C., Baracos, V., Guttridge, D., Jatoi, A., Kalantar-Zadeh, K., Lochs, H., Mantovani, G., Marks, D., Mitch, W. E., Muscaritoli, M., Najand, A., Ponikowski, P., Rossi Fanelli, F., Schambelan, M., Schols, A., Schuster, M., … Anker, S. D. (2008). Cachexia: A new definition. *Clinical Nutrition, 27*(6), 793–799. https://doi.org/10.1016/j.clnu.2008.06.013

Gunsar, F., Raimondo, M. L., Jones, S., Terreni, N., Wong, C., Patch, D., Sabin, C., & Burroughs, A. K. (2006). Nutritional status and prognosis in cirrhotic patients. *Alimentary Pharmacology & Therapeutics, 24*(4), 563–572. https://doi.org/10.1111/j.1365-2036.2006.03003.x

Haj Ali, S., Abu Sneineh, A., & Hasweh, R. (2022). Nutritional assessment in patients with liver cirrhosis. *World Journal of Hepatology, 14*(9), 1694–1703. https://doi.org/10.4254/wjh.v14.i9.1694

Henkel, A. S., & Buchman, A. L. (2006). Nutritional support in patients with chronic liver disease. *Nature Clinical Practice Gastroenterology & Hepatology, 3*(4), 202–209. https://doi.org/10.1038/ncpgasthep0443

Huang, H., Shen, H., Ning, K., Zhang, R., Sun, W., Li, B., Jiang, H., Wang, W., Du, J., Zhao, M., Yi, Z., Li, J., Zhu, R., Lu, S., Xie, S., Wang, X., Fu, W., Gao, C., & Hao, W. (2021). Quality of Life and Its Correlates in Alcohol Use Disorder Patients with and Without Depression in China. *Frontiers in Psychiatry, 11*, 627338. https://doi.org/10.3389/fpsyt.2020.627338

Huisman, E. J., Trip, E. J., Siersema, P. D., van Hoek, B., & van Erpecum, K. J. (2011). Protein energy malnutrition predicts complications in liver cirrhosis. *European Journal of Gastroenterology & Hepatology, 23*(11), 982–989. https://doi.org/10.1097/meg.0b013e32834aa4bb

Kalaitzakis, E., Simrén, M., Olsson, R., Henfridsson, P., Hugosson, I., Bengtsson, M., & Björnsson, E.

(2006). Gastrointestinal symptoms in patients with liver cirrhosis: Associations with nutritional status and health-related quality of life. *Scandinavian Journal of Gastroenterology, 41*(12), 1464–1472. https://doi.org/10.1080/00365520600825117

Lata, J., Husová, L., Juránková, J., Senkyrík, M., Díte, P., Dastych, M. Jr., Dastych, M., & Kroupa, R. (2006). Factors participating in the development and mortality of variceal bleeding in portal hypertension-possible effects of the kidney damage and malnutrition. *Hepatogastroenterology, 53*(69), 420-425.

McClain, C. J., Barve, S. S., Barve, A., & Marsano, L. (2011). Alcoholic Liver Disease and Malnutrition. *Alcoholism: Clinical and Experimental Research, 35*(5), 815–820. https://doi.org/10.1111/j.1530-0277.2010.01405.x

Merli, M., Lucidi, C., Giannelli, V., Giusto, M., Riggio, O., Falcone, M., Ridola, L., Attili, A. F., & Venditti, M. (2010). Cirrhotic Patients Are at Risk for Health Care–Associated Bacterial Infections. *Clinical Gastroenterology and Hepatology, 8*(11), 979-985.e1. https://doi.org/10.1016/j.cgh.2010.06.024

Mokdad, A. A., Lopez, A. D., Shahraz, S., Lozano, R., Mokdad, A. H., Stanaway, J., Murray, C. J., & Naghavi, M. (2014). Liver cirrhosis mortality in 187 countries between 1980 and 2010: a systematic analysis. *BMC Medicine, 12*(1). https://doi.org/10.1186/s12916-014-0145-y

Mondal, D., Das, K., & Chowdhury, A. (2022). Epidemiology of Liver Diseases in India. *Clinical Liver Disease, 19*(3), 114–117. https://doi.org/10.1002/cld.1177

Mukherjee, P. S., Vishnubhatla, S., Amarapurkar, D. N., Das, K., Sood, A., Chawla, Y. K., Eapen, C. E., Boddu, P., Thomas, V., Varshney, S., Hidangmayum, D. S., Bhaumik, P., Thakur, B., Acharya, S. K., & Chowdhury, A. (2017). Etiology and mode of presentation of chronic liver diseases in India: A multi centric study. *PLOS ONE, 12*(10), e0187033. https://doi.org/10.1371/journal.pone.0187033

Nixon, S. J., Garcia, C. C., & Lewis, B. (2023). Age as a potential modulator of alcohol-related deficits. *Alcohol, 107*, 12–18. https://doi.org/10.1016/j.alcohol.2022.07.004

Nixon, S. J., Torres, S. A., Boissoneault, J., Garcia, C. C., & Lewis, B. (2025). Neurobehavioral concomitants of alcohol use in older healthy adults. *Frontiers in Human Neuroscience,* 19. https://doi.org/10.3389/fnhum.2025.1693330

Nunes, G., Santos, C. A., Barosa, R., Fonseca, C., Barata, A. T., & Fonseca, J. (2017). Outcome and nutritional assessment of chronic liver disease patients using anthropometry and subjective global assessment. *Arquivos de Gastroenterologia, 54*(3), 225–231. https://doi.org/10.1590/s0004-2803.201700000-28

Paul, S., & Sadhukhan, G. (2023). Combinatorial impact of Chelerythrine and DADS in the restoration of liver physiology during carcinogenic exposure in mice (Mus musculus). *Int. J. Exp. Res. Rev.*, *34*(Special Vol.), 57-71. https://doi.org/10.52756/ijerr.2023.v34spl.007

Piekarski, D., Sullivan, E. V., Pfefferbaum, A., & Zahr, N. M. (2022). Poor subjective sleep predicts compromised quality of life but not cognitive impairment in abstinent individuals with Alcohol Use Disorder. *Alcohol, 103*, 37–43. https://doi.org/10.1016/j.alcohol.2022.07.001

Pillay, J., Gaudet, L. A., Saba, S., Vandermeer, B., Ashiq, A. R., Wingert, A., & Hartling, L. (2024). Falls prevention interventions for community-dwelling older adults: systematic review and meta-analysis of benefits, harms, and patient values and preferences. *Systematic Reviews, 13*(1). https://doi.org/10.1186/s13643-024-02681-3

Pimpin, L., Cortez-Pinto, H., Negro, F., Corbould, E., Lazarus, J. V., Webber, L., & Sheron, N. (2018). Burden of liver disease in Europe: Epidemiology and analysis of risk factors to identify prevention policies. *Journal of Hepatology, 69*(3), 718–735. https://doi.org/10.1016/j.jhep.2018.05.011

Plauth, M., & Schütz, E.T. (2002). Cachexia in liver cirrhosis. *International Journal of Cardiology, 85*(1), 83–87. https://doi.org/10.1016/s0167-5273(02)00236-x

Plauth, M., Cabré, E., Riggio, O., Assis-Camilo, M., Pirlich, M., Kondrup, J., Ferenci, P., Holm, E., vom Dahl, S., Müller, M. J., & Nolte, W. (2006). ESPEN Guidelines on Enteral Nutrition: Liver disease. *Clinical Nutrition, 25*(2), 285–294. https://doi.org/10.1016/j.clnu.2006.01.018

Rahimtoola, S. H. (2014). Foreword. *Current Problems in Cardiology, 39*(6), 157. https://doi.org/10.1016/j.cpcardiol.2014.02.003

Rao, K., Devi, J., Anuradha, Y., G, K., Kumar, M., & Rao, M. S. (2024). Enhancing Liver Disease Detection and Management with Advanced Machine Learning Models. *International Journal*

of Experimental Research and Review, *42*, 100-110. https://doi.org/10.52756/ijerr.2024.v42.009

Reccia, I., Kumar, J., Akladios, C., Virdis, F., Pai, M., Habib, N., & Spalding, D. (2017). Non-alcoholic fatty liver disease: A sign of systemic disease. *Metabolism, 72*, 94–108. https://doi.org/10.1016/j.metabol.2017.04.011

Rivera Irigoin, R., & Abilés, J. (2012). Soporte nutricional en el paciente con cirrosis hepática. *Gastroenterología y Hepatología, 35*(8), 594–601. https://doi.org/10.1016/j.gastrohep.2012.03.001

Sinclair, M., Gow, P. J., Grossmann, M., & Angus, P. W. (2016). Review article: sarcopenia in cirrhosis – aetiology, implications and potential therapeutic interventions. *Alimentary Pharmacology & Therapeutics, 43*(7), 765–777. https://doi.org/10.1111/apt.13549

Tessari, P. (2003). Protein metabolism in liver cirrhosis: from albumin to muscle myofibrils. *Current Opinion in Clinical Nutrition and Metabolic Care, 6*(1), 79–85. https://doi.org/10.1097/00075197-200301000-00012

Vieira, P.M., De-Souza, D.A., & Oliveira, L.C. (2013). Nutritional assessment in hepatic cirrhosis, clinical, anthropometric, biochemical and hematological parameters. *Nutr. Hosp., 28*(5), 1615-21.

Wani, M., Muzamil, A., Altaf, A., & Shaheen, F. (2024). Imaging Characteristics and Diagnostic Challenges of Alveolar Hydatid Disease in The Liver: A Comprehensive Analysis. *International Journal of Experimental Research and Review, 39*(Spl Volume), 170-179. https://doi.org/10.52756/ijerr.2024.v39spl.013

World Health Organization (2020). Newsroom Q & A Detail: Malnutrition. [Internet]. WHO; 2020 Apr 15. https://www.who.int/news-room/questions-and-answers/item/malnutrition

Yuan, J., Wu, C., Wu, L., Fan, X., Zeng, T., Xu, L., Wei, Y., Zhang, Y., Wang, H., Peng, Y., Kang, C., & Yang, J. (2022). The Association of P300 Components with Clinical Characteristics and Efficacy of Pharmacotherapy in Alcohol Use Disorder. *Frontiers in Psychiatry*, 13. https://doi.org/10.3389/fpsyt.2022.770714

How to cite this Article:

Anamitra Hait, Arbind Kumar Chaudhary and Avinash S.S. (2025). Alcohol as a Myotoxic Modifier of Nutritional Status: Distinct Nutritional Phenotypes in Alcoholic versus Non-Alcoholic Chronic Liver Disease. *International Journal of Experimental Research and Review*, *49*, 114-125.

DOI: https://doi.org/10.52756/ijerr.2025.v49.010

This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License.